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CymaBay
Transforming into a fully integrated company

CymaBay
• Focus on improving the lives of patients with inflammatory liver diseases
• Targeting indications with limited or no approved therapies and high unmet need
• Forward integrating into a Commercial organization to prepare for first launch

Seladelpar
• Poised to be the first and only selective PPARδ agonist approved for patients
• Potent PPARδ agonist with pleiotropic regulation of critical liver disease pathways
• Oral once daily dose with advantageous tolerability profile

Value
• Three late-stage development programs reading out in the next 2 years
• Highly de-risked pivotal Phase 3 in Primary Biliary Cholangitis (PBC) with Breakthrough Therapy Designation (BTD)
• Phase 2 initiated in Primary Sclerosing Cholangitis (PSC), a second orphan cholestatic liver disease
• NASH Phase 2b presents upside and strategic options
CymaBay
Delivering on seladelpar to drive value

2019

- NASH Ph 2 Enrolled
- PBC Breakthrough Therapy (FDA)
- CymaBay Raises $115M
- NASH Ph 2 Liver Fat 12 Weeks
- PBC Ph 3 ENHANCE Enrolled
- PBC Ph 2 First Patient
- PBC NDA Filed
- PBC Ph 3 Readout
- PSC Ph 2 Fully Enrolled
- NASH Ph 2 Histology 52 Weeks
- PBC Ph 2 Readout 52 Weeks

2020-21

First Launch

PBC Ph 3 Readout
Seladelpar
Differentiated opportunity addressing unmet needs in liver disease

1.8 Å RMSD X-ray Crystal Structure
Seladelpar - PPARδ Ligand Binding Domain
EC$_{50}$ = 2nM

First potent and selective PPARδ agonist in development for inflammatory liver diseases

Oral, once daily with clinical activity down to 5 mg and clinical experience with exposures beyond one year

Regulation of pathways important in inflammatory liver diseases; bile acids, lipid metabolism, inflammation and fibrosis
Seladelpar
Targets all important cell types in liver disease

Decrease Bile Acids
- ↓ Cholesterol synthesis
- ↓ Bile acid synthesis (C4)
- ↑ Transport

Anti-Fibrotic
- ↓ Connective Tissue Growth Factor
- ↓ Stellate cell activation
- ↓ Collagen synthesis/deposition

Anti-Inflammatory
- ↓ NFκB-dependent gene activation
- ↓ Inflammatory cytokines
- ↓ hs-C-Reactive Protein

Increase Lipid Metabolism
- ↓ Cholesterol/LDL-C
- ↑ Fatty acid oxidation
- ↑ Insulin sensitivity

Regulates genes that control pathways in liver health and disease
Potential to serve the two key unmet needs for patients with PBC –
better efficacy and improved tolerability
Primary Biliary Cholangitis
Orphan, autoimmune inflammatory disease of the liver

- Impairment of bile flow (cholestasis), portal inflammation and destruction of bile ducts
- Elevated serum markers of cholestasis including alkaline phosphatase (AP), gamma-glutamyl transferase (GGT) and total bilirubin
- Clinical symptoms of fatigue and pruritus (itching)
- Affects 1 in 1,000 women over 40 (~130,000 patients in the U.S.)

**AP below 1.67x the upper limit of normal and normal total bilirubin are clinical surrogates for slowing disease progression**
Despite Current Therapies Unmet Need Remains
*Patients need improved efficacy and better tolerability*

**Ursodeoxycholic Acid (UDCA)**

1st Line

▲ First line therapy for PBC
▼ ~40% inadequate responders: AP >1.67x ULN
▼ Additional 5% are intolerant to therapy

**Obeticholic Acid (Ocaliva)**

2nd Line

▲ Add-on therapy for UDCA inadequate responders
▲ Monotherapy for UDCA intolerant patients
▲ AP/bilirubin as biomarkers for accelerated approval
▼ ~50% inadequate responders
▼ Can cause or worsen pruritus

**Seladelpar is being developed as a potential improved 2nd line treatment for PBC**
Seladelpar Phase 2 Open Label Study in PBC
Add-on for patients with an inadequate response or intolerance to UDCA

**Entry Criteria:**
AP ≥ 1.67x ULN; ALT/AST ≤ 3x ULN; Total Bilirubin ≤ 2x ULN

<table>
<thead>
<tr>
<th>Dose Level</th>
<th>Treatment Duration</th>
<th>Option for Dose Adjustment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Seladelpar 5 mg</td>
<td>Week 12†</td>
<td>Seladelpar 5 mg or 10 mg qd</td>
</tr>
<tr>
<td>Seladelpar 10 mg</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Primary Outcome:**
% change in AP from baseline

**Secondary Outcomes:**
composite responder rate*, AP normalization, changes in liver, metabolic and inflammatory markers

†At week 12 a dose adjustment in the 5/10 mg group was made based on patient response and tolerability.
*Patients with AP≤1.67x ULN, ≥15% drop in AP from baseline and normal total bilirubin.
# Seladelpar Phase 2 Study in PBC

## Baseline demographics of mITT population (N=34 at 52 weeks)*

*Data as of July 23, 2018 and includes only patients that reached 52-weeks at such date

†Median [Quartiles: 25, 75]. mITT, modified intention to treat; VAS, visual analogue scale.

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Mean (SD)</th>
<th>Parameters (Reference Range)</th>
<th>Seladelpar 5/10 mg (n=17)</th>
<th>Seladelpar 10 mg (n=17)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, years</td>
<td>49 (5)</td>
<td></td>
<td>48 (11)</td>
<td></td>
</tr>
<tr>
<td>Female/male</td>
<td>17/0</td>
<td></td>
<td>16/1</td>
<td></td>
</tr>
<tr>
<td>History of Pruritus, n (%)</td>
<td>11 (65)</td>
<td></td>
<td>14 (82)</td>
<td></td>
</tr>
<tr>
<td>Pruritus VAS (0-100)</td>
<td>19 (22)</td>
<td></td>
<td>37 (31)</td>
<td></td>
</tr>
<tr>
<td>AP (37-116 U/L)</td>
<td>351 (166)</td>
<td></td>
<td>279 (74)</td>
<td></td>
</tr>
<tr>
<td>ALT (6-41 U/L)</td>
<td>41 (17)</td>
<td></td>
<td>52 (25)</td>
<td></td>
</tr>
<tr>
<td>Total bilirubin† (0.10-1.10 mg/dL)</td>
<td>0.56 [0.50, 0.70]</td>
<td></td>
<td>0.75 [0.57, 1.14]</td>
<td></td>
</tr>
<tr>
<td>UDCA Dose, mg/kg/day</td>
<td>15 (4)</td>
<td></td>
<td>17 (3)</td>
<td></td>
</tr>
</tbody>
</table>
Seladelpar Phase 2 Study in PBC
Rapid and sustained decrease in AP through week 52

Mean Percent AP Change Baseline to Week 52

Mean AP from Baseline to Week 52

*Doses adjusted for 5/10 mg group

Decreases in AP >45% observed at 5/10 mg and 10 mg

*P<0.0001 for both groups compared to baseline values
Mean ± SEM

Cymabag
Seladelpar Phase 2 Study in PBC
Up to 71% of patients achieved the composite efficacy endpoint

Composite Responder Rate

<table>
<thead>
<tr>
<th>Seladelpar</th>
<th>Responder Rate, %</th>
</tr>
</thead>
<tbody>
<tr>
<td>5/10 mg</td>
<td>59%</td>
</tr>
<tr>
<td>10 mg</td>
<td>71%</td>
</tr>
</tbody>
</table>

AP Normalization

<table>
<thead>
<tr>
<th>Seladelpar</th>
<th>AP Normalization, %</th>
</tr>
</thead>
<tbody>
<tr>
<td>5/10 mg</td>
<td>24%</td>
</tr>
<tr>
<td>10 mg</td>
<td>29%</td>
</tr>
</tbody>
</table>
Seladelpar Phase 2 Study in PBC
Average total bilirubin levels stable through week 52

Mean Total Bilirubin

Dose adjustment for 5/10 mg group
Seladelpar Phase 2 Study in PBC
Patient reported pruritus: Treatment not associated with increase

Visual Analog Scale (VAS) through week 52

Median Change in VAS

Baseline median VAS, 5/10 mg=10 and 10 mg=32

Dose adjustment for 5/10 mg group

- In patients with baseline itch, the median changes in VAS were -30% and -66% in the 5/10 mg and 10 mg groups, respectively

Median VAS

Dose adjustment for 5/10 mg group

5/10 mg, n=17
10 mg, n=17
Seladelpar Phase 2 Study in PBC
Significant decreases in transaminase through week 52

Median Percent ALT Change

Week

0 2 12 26 52

Median % Change

-50 -40 -30 -20 -10 0

Dose adjustment for 5/10 mg group

-31%

-33%

*P<0.0001, compared to baseline values

Mean ALT

5/10 mg, n=17
10 mg, n=17

Mean ALT, U/L

0 10 20 30 40 50 60

2 12 26 52

Dose adjustment for 5/10 mg group

ULN

Mean ± SEM
Seladelpar Phase 2 Study in PBC
Safety summary (n=119)

- 11 serious AEs in the study; none were deemed related to seladelpar
- No ≥ grade 3 ALT elevations
- Three discontinuations
  - Related: Grade 1 gastroesophageal reflux
  - Unrelated: Pneumonia & worsening of pruritus
- No discontinuations for transaminase elevations
- Overall, no increase in pruritus

<table>
<thead>
<tr>
<th>AE</th>
<th>5/10 mg</th>
<th>10 mg</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pruritus</td>
<td>22%</td>
<td>18%</td>
</tr>
<tr>
<td>Fatigue</td>
<td>13%</td>
<td>7%</td>
</tr>
<tr>
<td>Diarrhea</td>
<td>14%</td>
<td>6%</td>
</tr>
<tr>
<td>Nausea</td>
<td>14%</td>
<td>6%</td>
</tr>
</tbody>
</table>
Seladelpar for PBC
*Significantly de-risked Phase 3 program*

- Response of up to 71% of patients in registration endpoint
- No signal for drug-induced itch
- Phase 2 study enrolled with 104 patients beyond 52 weeks
- ENHANCE Phase 3 global registration study enrolling
ENHANCE Phase 3 Pivotal Study
Phase 3 study design

Primary Outcome: Composite responder rate (AP <1.67xULN, ≥15% decrease in AP, total bilirubin ≤ULN)

Secondary Outcomes: Proportion of patients with AP ≤1.0xULN at 6 and 12 months
Change from baseline in pruritus Numerical Rating Scale using e-diary at 6 months

n=240 (80/group)

Long Term Safety Study (CB8025-31731)
### ENHANCE Phase 3 Pivotal Study

**Same population, dose and endpoint as Phase 2 study**

<table>
<thead>
<tr>
<th>Population</th>
<th>Design</th>
<th>Primary Outcome</th>
<th>Secondary Outcomes</th>
</tr>
</thead>
</table>
| - Intolerance or inadequate response to UDCA  
- AP ≥ 1.67 x ULN, bilirubin ≤ 2 x ULN  
- Includes patients with severe pruritus | - Double blind, 52-week, placebo-controlled  
- Seladelpar 5/10 mg titration and 10 mg vs. placebo (1:1:1 randomization)  
- Stratified by AP and pruritus | - Composite responder rate (AP < 1.67 x ULN, ≥15% decrease in AP, total bilirubin ≤ ULN) | - Proportion of patients with AP ≤ 1.0 x ULN at 6 and 12 months  
- Change from baseline in pruritus Numerical Rating Scale using e-diary at 6 months |
Anti-cholestatic and Anti-inflammatory Activities that May Support Potential Treatment Effect in PSC
Primary Sclerosing Cholangitis
*Orphan, cholestatic inflammatory disease of the liver*

- Characterized by diffuse inflammation and fibrosis of both intra- and extra-hepatic bile ducts
- May progress to end-stage liver disease
- Common initial symptoms are fatigue, abdominal pain and itching (pruritus)
- ~70% of patients have IBD
- Cholangiocarcinoma develops in 8–30% of patients
- Affects men 2:1 (~40,000 patients in the U.S.)

*Only effective therapy is liver transplant*
Seladelpar PSC
24-Week Phase 2 study design

**Day 1**
- Seladelpar 5 mg PO QD (n=25)
- Seladelpar 10 mg PO QD (n=25)
- Seladelpar 25 mg PO QD (n=25)
- Placebo PO QD (n=25)

**Week 24**
- Placebo PO QD (n=25)
- Seladelpar 5 mg PO QD (n=25)
- Seladelpar 10 mg PO QD (n=25)
- Seladelpar 25 mg PO QD (n=25)

**Primary Outcome:** % change in AP from baseline to week 24

**Secondary Outcomes:** FibroScan, MRCP and other exploratory measures
Potential mechanism that decreases disease drivers of NASH:

• metabolic load – reduces bile acids, cholesterol & lipids
• cell stress and injury – reverses hepatocellular ballooning
• inflammation and fibrosis – lowers macrophages & collagen
Seladelpar for NASH
Potential role for PPARδ agonists in the treatment of NASH

Pathological Progression from NAFLD to NASH

- Steatosis
  - Insulin resistance
  - Bile acids
  - Free Cholesterol
  - Lipotoxic lipids

- ER stress/ROS
  - Inflammatory mediators

- Activation & recruitment
  - Kupffer cells
  - Macrophages
  - Neutrophils
  - Cell death
  - Stellate cell activation

- Extracellular matrix deposition & remodeling

Seladelpar (PPARδ) Pharmacology
Seladelpar Phase 2b Study in NASH
Paired-liver biopsy 52-week study design

Study design meets FDA/EMA guidance criteria to enable a Ph 3 program

- Seladelpar 10 mg PO QD (n=50)
- Seladelpar 20 mg PO QD (n=50)
- Seladelpar 50 mg PO QD (n=50)
- Placebo PO QD (n=25)

Secondary Outcome: Change in Histology at 52 weeks
Seladelpar Phase 2b Study in NASH
Enrolled patients reflective of phase 3 population

**Population**
- Histologically confirmed NASH at baseline
- Liver fat content (LFC) ≥10% by MRI-PDFF
- F1 to F3, NAS ≥ 4; 1 point in each component
- Includes diabetics and non-diabetics

**12-Week Outcome Measures**
- 12-week relative change in LFC
- Liver biochemistry: ALT, AST, GGT, AP
- Lipid markers: LDL-C, triglycerides
- Other inflammatory markers: hs-CRP

**Other Key Outcome Measures**
- Safety and tolerability
- 52-week histological improvement in NAS and fibrosis
- LFC and cT1 by LMS
- Liver stiffness by MRE and Fibroscan
- Biochemical fibrosis markers and Histoindex® quantitative digital pathology

*Study blinded and ongoing to 52 weeks*
## Baseline demographics and patient characteristics (mITT)

<table>
<thead>
<tr>
<th>Parameter (Mean ± SD)</th>
<th>Placebo (n = 26)</th>
<th>10 mg (n = 50)</th>
<th>20 mg (n = 47)</th>
<th>50 mg (n = 48)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (Years)</td>
<td>54 (10.5)</td>
<td>53 (12.6)</td>
<td>57 (12.0)</td>
<td>53 (11.3)</td>
</tr>
<tr>
<td>Male/Female (%)</td>
<td>30.8/69.2</td>
<td>30.0/70.0</td>
<td>31.9/68.1</td>
<td>33.3/66.7</td>
</tr>
<tr>
<td>Body Weight (kg)</td>
<td>104.4 (19.9)</td>
<td>95.3 (21.6)</td>
<td>100.7 (22.9)</td>
<td>99.9 (19.9)</td>
</tr>
<tr>
<td>MRI-PDFF (%)</td>
<td>22.3 (9.5)</td>
<td>22.0 (7.8)</td>
<td>20.8 (6.1)</td>
<td>20.5 (6.8)</td>
</tr>
<tr>
<td>ALT (U/L)</td>
<td>61.0 (34.7)</td>
<td>60.4 (29.6)</td>
<td>57.4 (26.3)</td>
<td>67.6 (40.2)</td>
</tr>
<tr>
<td>AST (U/L)</td>
<td>43.5 (24.5)</td>
<td>45.2 (24.9)</td>
<td>46.0 (21.1)</td>
<td>46.3 (27.9)</td>
</tr>
<tr>
<td>GGT (U/L)</td>
<td>99.3 (177.5)</td>
<td>84.7 (124.4)</td>
<td>97.4 (80.6)</td>
<td>66.5 (45.2)</td>
</tr>
<tr>
<td>AP (U/L)</td>
<td>82.1 (34.1)</td>
<td>83.9 (25.1)</td>
<td>81.1 (28.0)</td>
<td>76.5 (21.6)</td>
</tr>
<tr>
<td>NAS</td>
<td>5.3 (1.1)</td>
<td>5.2 (1.0)</td>
<td>5.1 (1.0)</td>
<td>5.1 (1.0)</td>
</tr>
<tr>
<td>Fibrosis Stage</td>
<td>2.1 (0.65)</td>
<td>2.1 (0.70)</td>
<td>2.3 (0.72)</td>
<td>2.1 (0.65)</td>
</tr>
<tr>
<td>LDL-C (mg/dL)</td>
<td>114.2 (45.5)</td>
<td>103.8 (33.0)</td>
<td>111.0 (47.6)</td>
<td>106.7 (40.0)</td>
</tr>
<tr>
<td>Triglycerides (mg/dL)</td>
<td>151.2 (51.3)</td>
<td>166.4 (79.5)</td>
<td>173.4 (72.8)</td>
<td>154.2 (93.8)</td>
</tr>
</tbody>
</table>

mITT = modified intent-to-treat population
Seladelpar Phase 2b Study in NASH
Changes in relative liver fat content by MRI-PDFF at 12 weeks

Comparative Relative Change from Baseline

Proportion of Subjects with > 30% Relative Change from Baseline

Mean Relative Change in LFC (%)

Proportion of Subjects (%)

p-values relative to placebo
Seladelpar Phase 2b Study in NASH
Positive changes in absolute and relative ALT over 12 weeks

**Change in Relative ALT Over Time**

- Placebo (n=27)
- 10 mg (n=53)
- 20 mg (n=51)
- 50 mg (n=50)

**Change in Absolute ALT Over Time**

- Placebo (n=27)
- 10 mg (n=53)
- 20 mg (n=51)
- 50 mg (n=50)
Seladelpar Phase 2b Study in NASH
Positive changes in absolute and relative GGT
### Seladelpar Phase 2b Study in NASH

*Dose dependent decreases in markers of hepatic injury at 12 weeks*

<table>
<thead>
<tr>
<th>%, LS Mean (SE)</th>
<th>Placebo (n = 27)</th>
<th>10 mg (n = 53)</th>
<th>20 mg (n = 51)</th>
<th>50 mg (n = 50)</th>
</tr>
</thead>
<tbody>
<tr>
<td>ALT</td>
<td>-8.9 (5.1) p=0.08</td>
<td>-22.9 (3.8) p&lt;0.0001</td>
<td>-32.0 (4.0) p&lt;0.0001</td>
<td>-37.5 (4.0) p&lt;0.0001</td>
</tr>
<tr>
<td>AST</td>
<td>-12.9 (5.8) p=0.03</td>
<td>-11.6 (4.4) p=0.009</td>
<td>-15.2 (4.5) p=0.001</td>
<td>-17.3 (4.5) p=0.0002</td>
</tr>
<tr>
<td>GGT</td>
<td>-4.5 (4.3) p=0.3</td>
<td>-28.2 (3.2) p&lt;0.0001</td>
<td>-37.6 (3.3) p&lt;0.0001</td>
<td>-43.1 (3.4) p&lt;0.0001</td>
</tr>
<tr>
<td>AP</td>
<td>4.4 (2.9) p=0.12</td>
<td>-19.1 (2.1) p&lt;0.0001</td>
<td>-25.1 (2.2) p&lt;0.0001</td>
<td>-33.4 (2.2) p&lt;0.0001</td>
</tr>
</tbody>
</table>

ALT, AST, GGT and AP data from safety population; p-values relative to baseline
Seladelpar Phase 2b Study in NASH
Positive changes in lipid and inflammation parameters at 12 weeks

LDL-C, TG and hs-CRP data from safety population
Seladelpar Phase 2b Study in NASH
Safety summary

- Majority of treatment emergent adverse events were mild to moderate and deemed unrelated to study drug
- The most common (>5%) treatment emergent adverse events included nausea, constipation, dizziness, headache, gastroesophageal reflux disease and upper abdominal pain
- Two SAEs both deemed unrelated to study drug
- No Grade 3 or greater ALT/AST elevations
## CymaBay Accomplishments and Goals

**Improving the lives of patients with liver diseases**

<table>
<thead>
<tr>
<th>Year</th>
<th>Accomplishments and Goals</th>
</tr>
</thead>
</table>
| 2019 | - Breakthrough Therapy Designation  
     |   - 12-week Ph 2b NASH data  
     |     - Initiate Ph 2 PSC study – Q3  
     |     - Complete enrollment of ENHANCE – Q4 |
| 2020 | - Ph 2 PBC full data – Q1  
     |   - 52-week Ph 2b NASH data (histology) – Q2  
     |   - Complete 52-week treatment period of ENHANCE – H2 |
| 2021 | - ENHANCE data – H1  
     |   - PSC Ph2 data – H2  
     |   - NDA filing PBC – H2 |